EXHIBIT B

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Alan Brayton
Brayton-Purcell, LLP
222 Rush Landing Road
P. O. Box 6169
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RE: BARRY KELLY

Dear Mr. Brayton:

At your request, I have reviewed additional information regarding Mr. Kelly. Mr. Kelly was previously seen by me on November 21, 2011. I sent my report to your office dated December 12, 2011.

Report of Dr. Sam Hammar dated April 23, 2012
Dr. Hammar reviewed interrogatories. He reviewed limited medical records. He reviewed my report dated December 12, 2011. Dr. Hammar reviewed deposition testimony of Mr. Kelly.

Dr. Hammar reviewed a slide from Baylor All Saints Medical Center, Fort Worth, Texas from 2011. The tissue was composed of neoplastic epithelioid cells that formed glandular structures. There was a fibrous stroma associated with the epithelioid cells.

Dr. Hammar concluded Mr. Kelly had peritoneal epithelioid mesothelioma caused by exposure to asbestos.

Review of Medical Records

Records of Baylor All Saints Medical Center
A CT-guided omental biopsy was performed on March 30, 2011.

I have reviewed the pathology report dated March 30, 2011, from the biopsy. It demonstrated fibrous tissue with a proliferation of epithelioid cells arranged in small clusters and partially having a glandular or pseudoglandular arrangement. The cells had a haphazard infiltrative pattern. Immunohistochemical stains were done. Calretinin and CK 5/6 were positive. MOC 31 and TAG72 were negative. CEA was negative. The slides

were interpreted as showing an epithelioid neoplasm consistent with malignant mesothelioma.

Records of Dr. Hisham Bismar
Dr. Bismar is in practice in Fort Worth, Texas.

Mr. Kelly was seen on December 9, 2010, because of a chronic cough for 16 weeks following a viral illness. He was not coughing up sputum. He was not short of breath. The albuterol inhaler helped relieve the cough somewhat. Spirometry was done that day and was normal. The FEV1 was 2.2 L. A chest x-ray done that day was also normal. He had no history of smoking. On examination, there were diminished breath sounds in the chest. The abdomen was soft and nontender. There was no organomegaly. There was no cyanosis, clubbing or edema. He was diagnosed with a cough from a viral illness. He was placed on Dulera and a tapering course of prednisone.

He was seen on December 29, 2010, complaining of feeling pressure in his chest. He had no fever or chills. On examination, the oxygen saturation was 97%. There were diminished breath sounds in the chest. There was no cyanosis, clubbing or edema. He was told to stop steroids. The chest CT scan showed nonspecific findings in the chest. There was mesenteric edema. An abdominal CT scan was ordered.

Mr. Kelly was seen on January 11, 2011. He denied having fever, chills, aches or weight changes. He was not short of breath and was not coughing. An abdominal CT scan dated January 5, 2011, showed ascites and edema in the mesenteric area. A chest x-ray on December 9, 2010, was clear. On examination, the oxygen saturation was 95% room air. There were diminished breath sounds in the chest. Cardiac examination was normal. The abdomen was soft and nontender. There was no organomegaly. He was diagnosed with having a cough related to a viral illness which was resolved. He was continued on Dulera. He was being referred for further evaluation of the ascites

Mr. Kelly was seen on April 28, 2011. He denied having shortness of breath or cough. He had no fever, chills, aches or weight changes. He denied having any chest pain. He was not having abdominal pain, nausea, vomiting or constipation. He had malignant peritoneal mesothelioma. He also had reactive airways disease, diabetes, hypertension, hypothyroidism, diverticulitis, and hyperlipidemia. Spirometry in December 2010 was normal. A chest x-ray on April 28, 2011, was described as being clear. He had no history of smoking. Mr. Kelly was on Byetta, carvedilol, Lasix, glimepiride, Lansoprazole, levothyroxine, Micardis, Prandin, pravastatin, and Precose. The oxygen saturation was 98% room air. There were diminished breath sounds in the chest. There were no wheezes. The abdomen was soft and nontender. There was no organomegaly. There was no cyanosis, clubbing or edema. The physician concluded that the reactive airways disease was resolved and the plan was to stop Dulera. He was awaiting evaluation at MD Anderson for the peritoneal mesothelioma.

Records of Dr. Mike Bismar
Dr. Bismar is a gastroenterologist.

Mr. Kelly was seen on January 11, 2011, because of ascites. He denied having any abdominal pain. He reported bloating and fatigue. His wife reported noticing a change in his abdomen over the last three to four months along with muscle wasting. His weight fluctuated between 180 and 198 pounds. He had colonoscopy in 2007 which was unremarkable. He had upper GI endoscopy in December 2009 which demonstrated gastritis. He complained of a cough. A chest CT scan demonstrated ascites. A CT scan of the abdomen and pelvis was then ordered. His omentum was thickened. His father had kidney cancer. On examination, he weighed 198 pounds, blood pressure 137/85, pulse 114, respirations 16. The lungs were clear. Cardiac examination was normal. The abdomen was soft and nontender. It was not distended. No ascites was identifiable on physical examination. There was no organomegaly. Lab work was done on January 5, 2011. The BUN was 11 and the creatinine was 2.1. The cause of the ascites was unclear. Liver function tests were ordered. A paracentesis was recommended. Referral to nephrology was recommended in view of the fact that the creatinine was elevated.

I have reviewed a letter dated January 13, 2011. The white blood count in the ascites was elevated at 82,000. The protein was 1.4. CEA was 1.2. Cytology did not reveal revealed malignant cells. The plan was to refer Mr. Kelly to Dr. Herr for further evaluation of his renal failure.

I have reviewed the charges for care. The charge for the original consultation was $538. The payment was $174. The charge for the second visit was $174. The payment was $53.

Records of Liver Consultants of Texas
Mr. Kelly was seen on May 11, 2012. Mr. Kelly was 60 years of age. He was seen in the past because of ascites. He was diagnosed with mesothelioma. He received treatment at the University of Chicago and was followed closely at the University of Texas Southwestern. He was first treated with chemotherapy and had significant side effects from the chemotherapy. He had pancytopenia. He was then switched to a gemcitabine-based regimen. He was off chemotherapy since November 2011. He was having periodic CT scans. Reportedly, his tumor responded to therapy and shrunk in size. He was undergoing paracenteses at this office as needed. The last one was in July 2011. He had one more paracentesis in September 2011 during hospitalization at the University of Chicago. He recently developed abdominal distention and fullness. He was not having abdominal pain. He denied having fever, chills, or shakes. His appetite was fair. He gained weight recently. He was on Byetta, carvedilol, levothyroxine, amlodipine, and Lansoprazole. On examination, the lungs were clear. Cardiac examination was normal. The abdomen was soft and nontender. There was no clinically significant free fluid. Bowel sounds were present. There was no organomegaly. There was no peripheral edema. A bedside ultrasound was performed. There was a thin layer of fluid in the left lower quadrant. The physician concluded that he had mesothelioma. He received

chemotherapy and responded to that therapy. He did not have significant ascites. A paracentesis was not recommended.

Records of the University of Texas Southwestern Medical Center
A chemistry panel was obtained in July 18, 2011. The sodium was 139, potassium 4.9, chloride 105, CO2 24, BUN 24, creatinine 1.84, glucose 196, calcium 8.8, albumin 3.3, protein 6.4, bilirubin 0.2, alkaline phosphatase 51, SGOT 20.

Mr. Kelly was seen by Dr. Dowell on September 22, 2011. Mr. Kelly was 60 years of age. He complained of mild fatigue. He was hospitalized at the University of Chicago following the third cycle of chemotherapy. Upon arrival at the University of Chicago, he had severe thrombocytopenia and neutropenia and was admitted. Prior to discharge he developed shortness of breath and an episode of hypoxemia. He had a CT angiogram which did not show pulmonary emboli. There was no evidence of deep venous thrombosis. He was started on low molecular heparin for a few days because of the question of pulmonary hypertension. Bronchoscopy was performed as well. Cultures were negative. The shortness of breath and hypoxia resolved. While in the hospital an abdominal CT scan which showed stable disease. A paracentesis was performed and 3 L of fluid was removed. The cytology was positive for mesothelioma. On examination, the blood pressure was 121/77, pulse 81, respirations 18, weight 175 pounds. The chest was clear. The abdomen was soft and nontender. The abdomen was not distended. There was no organomegaly. There was a positive fluid wave. There was no cyanosis, clubbing or edema. A CBC was obtained. The white blood count was 6600, hemoglobin 10.5, platelet count 389,000. Liver function tests were normal. The creatinine was 2.1. The conclusion was that he got some benefit from chemotherapy but he was having worsening renal function, so he was not considered a candidate for additional Alimta. Dr. Dowell spoke with Dr. Kindler in Chicago. The plan was to proceed with carboplatin and gemcitabine.

Mr. Kelly was next seen by Dr. Dowell on October 17, 2011. He received cycle one of gemcitabine and carboplatin three weeks ago. He was unable to get day eight of gemcitabine because of cytopenia. He also received a transfusion because of progressive anemia. He was currently asymptomatic other than mild fatigue. The blood pressure was 133/72, pulse 72, respirations 18, weight 179 pounds, height 6 feet. He had persistent neutropenia.

A chemistry panel was obtained on October 17, 2011. The sodium was 139, potassium 4.8, chloride 105, CO2 25, BUN 49, creatinine 2.22, glucose 68, calcium 9.6, albumin 4.0, protein 6.9, bilirubin 0.5, alkaline phosphatase 71, SGOT 27.

A CBC was obtained on October 17, 2011. The white blood count was 5300, hemoglobin 10.9, hematocrit 31.4, platelet count 252,000.

A chemistry panel was obtained on November 8, 2011. The sodium was 140, potassium 4.7, chloride 108, CO2 26, BUN 35, creatinine 2.36, glucose 63, calcium 8.7, albumin 3.8, protein 6.6, bilirubin 0.3, alkaline phosphatase 65, SGOT 25.

A CBC was obtained on November 8, 2011. The white blood count was 3700, hemoglobin 9.1, hematocrit 25.7, platelet count 235,000.

A CBC was obtained on November 15, 2011. The white blood count was 1600, hemoglobin 8.2, hematocrit 23.6, platelet count 95,000.

Mr. Kelly was seen on November 11, 2011. The second cycle of chemotherapy was delayed by one week. He received carboplatin and gemcitabine. He was again unable to receive day eight treatment because of thrombocytopenia. The platelet count was 92,000. He was now being seen to determine whether he should get cycle three of treatment. He was continuing to do fairly well. He did not require another paracentesis since he returned from Chicago. He was doing fairly well. He thought that there may be slightly more fluid in his abdomen. The blood pressure was 132/78, pulse 78, respirations 18, weight 180 pounds. The lungs were clear. The abdomen was soft and nontender. It was not distended. The creatinine was 2.3. The white blood count was 3700, hemoglobin 9.1, platelet count 235,000. The plan was to reduce the dose of gemcitabine further and to continue carboplatin. The plan was to give him chemotherapy once he returned from Chicago.

Mr. Kelly was seen by Dr. Dowell on January 24, 2012. It was pointed out that he received two additional cycles of treatment. The decision was to stop treatment at that point and have him seen at the University of Chicago. Scanning showed stable disease. He was continuing to do reasonably well. His strength was somewhat improved off chemotherapy. He noticed an increase of about ten pounds over the past few months. He did not feel like he needed a paracentesis. He had no fever. On examination, the blood pressure was 116/68, pulse 68, respirations 18, weight 188 pounds. The lungs were clear. The abdomen was protuberant. There was a positive fluid wave. There was no organomegaly. There was no cyanosis, clubbing or edema. They had a discussion about the fact that there was no standard second line therapy for his disease. He was not a candidate for experimental studies because of the renal insufficiency. He was scheduled to see Dr. Kindler in followup in early March with repeat scans.

A chemistry panel was obtained on January 24, 2012. The sodium was 141, potassium 4.8, chloride 105, CO2 25, BUN 33, creatinine 2.37, glucose 117, calcium 9.4, albumin 4.1, protein 7.0, bilirubin 0.3, alkaline phosphatase 83, SGOT 19.

A CBC was obtained on January 24, 2012. The white blood count was 6200, hemoglobin 10.0, hematocrit 29, platelet count 249,000.

Mr. Kelly was seen by Dr. Dowell on April 24, 2012. Mr. Kelly complained of abdominal swelling. His clinical course was reviewed. Mr. Kelly developed increasing abdominal girth in November 2010 and was found to have ascites. A biopsy was done in March 2011. He was found to have an epithelioid neoplasm consistent with mesothelioma. The pathology was reviewed at MD Anderson. He was then seen by several physicians including the surgery group at MD Anderson. They felt he was not a good candidate for maximal tumor debulking and hyperthermic chemotherapy because of underlying renal insufficiency. He was referred to Dr. Kindler at the University of Chicago who recommended chemotherapy with carboplatin and Alimta or carboplatin and gemcitabine. It was elected to give him carboplatin and Alimta. He received three cycles and returned to the University of Chicago. The second cycle was delayed one week because of anemia requiring a transfusion. Following the third cycle, he was seen at the University of Chicago where he had severe thrombocytopenia and neutropenia. He also had epistaxis and was admitted to the hospital. He developed some shortness of breath and hypoxemia. He was not thought to have a pulmonary embolus. He had pneumonia or interstitial pneumonitis related to the Alimta. A CT scan showed stable disease. He had a paracentesis performed. The cytology was positive for mesothelioma. He returned to Texas and elected to continue carboplatin and gemcitabine. He had two cycles, but was unable to receive day eight chemotherapy because of cytopenia. The chemotherapy was then stopped.

He was recently seen at the University of Chicago and was found to have stable disease. He was continuing to do well. He was active. He noted some weight gain and some increased swelling in his abdomen presumably due to increasing ascites. His last paracentesis was in August. The blood pressure was 130/73, pulse 76, respirations 18, height 6 feet, weight 193 pounds. There was no adenopathy. The lungs were clear. Cardiac examination was normal. The abdomen was firm and distended with a positive fluid wave. The abdomen was not tender. There was no cyanosis or clubbing. There was trace edema below the knee bilaterally. He was diagnosed with peritoneal mesothelioma with stable disease and he was doing quite well. He was having slowly increasing ascites. Dr. Dowell thought that he would need a paracentesis. He had that done locally by a liver specialist closer to home. He continued to have mild hyperkalemia and renal insufficiency which was stable. Followup in six weeks was recommended and then again in three months.

A CBC was obtained on April 24, 2012. The white blood count was 6600, hemoglobin 10.6, hematocrit 30.5, platelet count 222,000.

A chemistry panel was obtained on April 24, 2012. The sodium was 140, potassium 4.8, chloride 107, CO2 23, BUN 37, creatinine 2.65, glucose 91, calcium 8.6, albumin 3.9, protein 6.7, bilirubin 0.4, alkaline phosphatase 75, SGOT 17.

A CBC was done on June 5, 2012. The white blood count was 5600, hemoglobin 10.8, hematocrit 31.0, platelet count 184,000.

A chemistry panel was obtained on June 5, 2012. The sodium was 136, potassium 4.4, chloride 104, CO2 25, BUN 38, creatinine 2.47, glucose 117, calcium 9.1, albumin 3.8, protein 6.4, bilirubin 0.5, alkaline phosphatase 69, SGOT 18.

Records of Dr. Manjushree Gautam

Mr. Kelly was seen on July 16, 2012. He had known mesothelioma and had paracenteses in the past. The last one done in their office was in July 2011. He complained of some abdominal distention/fullness recently. He had no associated abdominal pain. On examination, he was 72 inches, 197 pounds, pulse 73, respirations 16, blood pressure 127/75. The lungs were clear. The abdomen was soft and nontender. There was no organomegaly. There was no clinically significant free fluid. There was no peripheral edema. A bedside ultrasound was performed. Mr. Kelly was noted to have a very small pocket of fluid in the left lower quadrant. A paracentesis was not recommended. He was on Byetta, carvedilol, levothyroxine, amlodipine, and Lansoprazole.

Records of Harris Methodist Southwest

I have reviewed an admission note dated April 30, 2007. Mr. Kelly was 55 years of age. He was seen because of the second postoperative visit for a ventral hernia repair. He complained of right-sided abdominal pain and fever at home. An abdominal CT scan showed stranding in the area of the ascending colon and a possible mass in the spleen. He appeared in the office pale and uncomfortable and was admitted. He had a history of non-insulin-dependent diabetes and hypertension. He was on Lasix, Prevacid, Precose, lisinopril, Pravachol, Prandin, Synthroid, Glucophage, and Byetta. On examination, the lungs were clear. Cardiac examination was normal. There was tenderness in the right upper quadrant with fullness. Bowel sounds were normal. The white blood count was 9100. Hematocrit was 35.8. The creatinine was 2.6 and the BUN was 36. He was diagnosed with abdominal pain and renal insufficiency.

He was seen on April 30, 2007, by a nephrologist. He was being admitted because of abdominal pain. On admission the BUN was 36 and the creatinine was 2.6. He was able to eat and drink despite the abdominal pain. On examination, the blood pressure was 131/75, pulse 74, respirations 20. The chest was clear. Abdomen was soft but the right side of his abdomen was very tender. Bowel sounds were active. He had renal insufficiency. He had a prior history of proteinuria suggesting the presence of a diabetic nephropathy. The baseline BUN and creatinine were not known at the time of the dictation.

I have reviewed a discharge summary regarding admission from April 30 to May 3, 2007. Mr. Kelly was 55 years of age. He presented with abdominal pain involving the right side. A CT scan showed an inflammatory process on the right side of the colon. This was thought to be either diverticulitis or a retrocecal appendix. He was treated with antibiotics. He was seen in the hospital because of renal insufficiency. He had a decrease in pain with antibiotics. He was continued on home intravenous antibiotics.

A CT scan of the abdomen and pelvis was obtained on June 1, 2007. Compared to the scan dated May 1, 2007, the bowel wall thickening and surrounding mesenteric edema of the ascending colon resolved. There was no evidence of bowel wall thickening involving the large or small bowel. The lower lung fields were unremarkable. The pancreas was not enlarged. Both adrenal glands and kidneys were unremarkable.

Records of Huguley Memorial Medical Center in Fort Worth, Texas
A chemistry panel was obtained on May 15, 2005. The sodium was 138, potassium 4.1, chloride 105, CO2 30, glucose 84, BUN 22, creatinine 1.7, calcium 8.1.

A CT scan of the abdomen and pelvis was obtained on May 17, 2005. There was a tiny right pleural effusion. There was diffuse inflammatory change involving the abdomen and pelvis. There was a small amount of free fluid in the pelvis. The inflammatory change around the sigmoid was improved.

A chemistry panel was obtained on May 20, 2005. The sodium was 140, potassium 4.6, chloride 103, CO2 31, glucose 104, BUN 20, creatinine 1.5, calcium 9.0.

I have reviewed a discharge summary dated May 20, 2005. Mr. Kelly was 53 years of age. He was admitted to the hospital with diverticulitis. He failed outpatient medical therapy. He was admitted for intravenous antibiotics. He was discharged in good condition.

I have reviewed an operative report dated March 23, 2007. A repair of a ventral hernia was performed.

I have reviewed an operative report dated August 8, 2007. A laparoscopic appendectomy was performed.

A chest film was obtained on December 4, 2007. It was normal.

A CT scan of the abdomen and pelvis was done on December 4, 2007. There was a focal splenic hypodensity. There was no evidence of hydronephrosis. There were no calcified gallstones. There was a small amount of free fluid present. There were scattered colonic diverticula without evidence of acute diverticulitis. There was nonspecific fat stranding involving the greater omentum in the right upper quadrant.

A CBC was obtained on December 4, 2007. White blood count was 12,000, hemoglobin 13.0, hematocrit 40.3, platelet count 404,000.

A chemistry panel was obtained on December 4, 2007. The sodium was 135, potassium 4.6, chloride 100, CO2 29, glucose 166, BUN 21, creatinine 2.0, calcium 8.9, alkaline phosphatase 62, amylase 92, lipase 60, SGOT 11, bilirubin 0.9.

An abdominal CT scan was performed on January 4, 2008. Linear radiopaque densities involving the posterior cecal wall were seen adjacent to the cecum, which were unchanged. These were findings compatible with a prior appendectomy. There were several rounded slightly hyperdense structures scattered within the stomach and the small bowel. It was unclear what this represented.

A hepatobiliary scan was performed on January 21, 2010. It was normal.

A chemistry panel was obtained on July 13, 2011. The sodium was 139, potassium 5.3, chloride one await, CO2 26, glucose 205, BUN 25, creatinine 1.8, calcium 8.6.

A CBC was obtained on July 13, 2011. The white blood count was 1100, hemoglobin 9.8, hematocrit 30.4, platelet count 163,000.

I have reviewed an operative report dated July 15, 2011. A central line was inserted.

Records of MD Anderson Cancer Center

I have reviewed a pathology report dated April 20, 2011. The tissue from the biopsy dated March 30, 2011, was reviewed. It demonstrated an epithelioid neoplasm consistent with malignant mesothelioma.

I have reviewed a note dated May 5, 2011. Mr. Kelly was 59 years of age. He developed ascites and pleural effusions in December 2010. He underwent several paracenteses without a definite diagnosis. He underwent a CT-guided omental biopsy which was consistent with epithelioid mesothelioma. Imaging studies were done on May 4, 2011. The CT of the abdomen demonstrated significant peritoneal disease in the subhepatic space with perihepatic fluid and tumor. There was a large omental cake. There were also serosal implants on the small bowel. The scan was unchanged from the April scan. It was pointed out that he had a significant history of renal disease with a baseline creatinine between 1.5 and 2. There was significant renal toxicity from cisplatin. Cisplatin was contraindicated for hyperthermic intraperitoneal chemotherapy. They discussed other options including using mitomycin-C but there was much less data regarding its efficacy for mesothelioma. Mitomycin C had significantly less renal toxicity although it was associated with some renal toxicity. Overall the physician felt that cytoreductive surgery with hyperthermic intraperitoneal chemotherapy was contraindicated in this setting. They discussed the possibility of chemotherapy.

Records of the University of Chicago

A CT scan of the chest was obtained on August 30, 2011. There was ground glass opacity within the right middle lobe and the lower lobes bilaterally, right greater than left. There was mild basilar septal thickening. There were a few peripheral peribronchial irregular nodules in the right middle lobe lateral segment. There were trace pleural fluid collections bilaterally, left greater than right.

Chest films were obtained on August 30, 2011. There was elevation of the right hemidiaphragm. There were linear opacities at the lung bases consistent with atelectasis.

I have reviewed a procedure note dated September 2, 2011. Bronchoscopy was performed. No endobronchial lesions were identified. There were no secretions. Bronchoalveolar lavage was performed in the right middle lobe.

I have reviewed a discharge summary dated September 3, 2011. Mr. Kelly was 59 years of age. He had peritoneal mesothelioma. He had received three cycles of chemotherapy. The last dose was on August 11, 2011. He presented with acute renal failure and pancytopenia. He had an episode of epistaxis that resolved within a day. He had diabetes, hypercholesterolemia, chronic kidney disease, hypertension, hypothyroidism, history of appendectomy, and history of hernia repair. He was an engineering officer in the Navy in the 1970s and worked primarily in shipyards. He was currently working as a systems engineer for a private company. On examination, the lungs were clear. The abdomen was distended with a fluid wave. He was diagnosed with peritoneal mesothelioma. He had acute kidney injury superimposed on chronic kidney disease attributed to the recent administration of an iodinated contrast for a staging CT plus possible volume depletion. He was hydrated. He had pancytopenia secondary to Alimta. The neutropenia was treated. He was given blood transfusions. On the fifth hospital day, he developed shortness of breath and cough productive of blood-tinged sputum. He required supplemental oxygen. There was suspicion that he had a pulmonary embolus and he was anticoagulated with Lovenox. The ventilation perfusion scan showed low probability of an embolus. Lower extremity Doppler studies were negative for deep venous thrombosis. He had a noncontrast CT scan of the chest which was suspicious for early bronchopneumonia. He was treated for pneumonia with moxifloxacin. Bronchoscopy with bronchoalveolar lavage was done. The blood pressure was poorly controlled during the admission and the dose of carvedilol was increased. Amlodipine was added. He was discharged on albuterol, amlodipine, Robitussin, folic acid, carvedilol, Byetta, Amaryl, Prevacid, Synthroid, and Pravachol.

A CT scan of the chest, abdomen and pelvis was obtained on December 14, 2011. There was unchanged linear basilar scarring in the lungs. There were scattered small mediastinal lymph nodes in the mediastinum. There was no adenopathy in the abdomen. There was a moderate amount of ascites. There was diffuse stranding of the peritoneal fat. The thickness of the omental cake at the level of the IMA measured 1.4 cm. It previously measured 1.6 cm. At the level of the umbilicus, it measured 1.1 cm. It previously measured 1.2 cm. There was scattered colonic diverticulosis without evidence of diverticulitis.

A CBC was obtained on December 14, 2011. The white blood count was 5400, hemoglobin 10.5, hematocrit 31.8, platelet count 237,000.

A chemistry panel was obtained on December 14, 2011. The glucose was 100, sodium 141, potassium 5.0, chloride 107, CO2 23, BUN 40, creatinine 2.6, calcium 9.1, bilirubin 0.4, albumin 4.2, alkaline phosphatase 83, SGOT 28.

I have reviewed a written note dated December 14, 2011. The note is very difficult to read. Mr. Kelly had peritoneal mesothelioma. He received Alimta and carboplatin and developed renal insufficiency. He was changed to gemcitabine and carboplatin and still had difficulty with myelosuppression. On examination he weighed 180 pounds. There were diminished breath sounds in the chest. A CT scan was interpreted as demonstrating a "minor response". It was recommended that further chemotherapy be held. It was further recommended that he have a CT scan in three months. He was diagnosed with mesothelioma, renal insufficiency, diabetes, and ascites.

Diagnoses

1. Peritoneal mesothelioma
2. Pancytopenia secondary to chemotherapy
3. Diabetes mellitus
4. Chronic kidney disease
5. History of hyperlipidemia
6. History of hypertension
7. Hypothyroidism
8. History of diverticulitis

Discussion

I will review Mr. Kelly's clinical course, which is also summarized in my report of December 12, 2011. At that time I did a history and physical on Mr. Kelly, reviewed medical records, and dictated a complete report.

Mr. Kelly had some coughing and aching beginning in August 2010. He was given an antibiotic but did not get better. At the end of 2010, he was referred to a pulmonologist. A CT scan of the chest demonstrated a small left pleural effusion. Ascites was also identified as well as a thickened and edematous cecum. He then had an abdominal and

pelvic CT scan performed which confirmed the presence of ascites. There was also soft tissue stranding and thickening of the omentum.

In January 2011, an ultrasound-guided paracentesis was performed. No malignant cells were identified. He was then sent to a liver specialist. No specific diagnosis was made.

In February 2011, he had a second paracentesis performed. No diagnosis was made.

On March 30, 2011 a CT-guided omental biopsy was performed and he was diagnosed with peritoneal mesothelioma. He was then referred to an oncologist who discussed therapeutic options including chemotherapy. It was recommended that he be seen at MD Anderson Cancer Center. The diagnosis of mesothelioma was confirmed by Dr. Sam Hammar and Dr. Nelson Ordonez.

In early May 2011, he was seen at MD Anderson. The omental biopsy was reviewed and the diagnosis of peritoneal mesothelioma was confirmed. The physicians did not think he was a candidate for debulking surgery with heated intraoperative chemotherapy because he had chronic kidney disease. It was recommended that he be seen by Dr. Hedy Kindler at the University of Chicago.

Mr. Kelly was seen in early June 2011 by Dr. Kindler who recommended chemotherapy with carboplatin and Alimta. Mr. Kelly indicated that he wanted to be treated closer to home. He was referred to Dr. Jonathan Dowell at the University of Texas Southwestern Medical Center for treatment.

Mr. Kelly was seen by Dr. Dowell and begun on chemotherapy with carboplatin and Alimta later in June 2011. He developed fatigue with the first course of chemotherapy. He had the second course of chemotherapy in July 18, 2011, and the third course on August 11, 2011. He developed some nausea, mouth sores, and fatigue with the chemotherapy.

Mr. Kelly then went back to the University of Chicago for re-evaluation. He drove from Texas to Chicago. During the drive, he developed epistaxis. When he arrived at the University of Chicago, he was found to be severely pancytopenic and was hospitalized. He received blood and platelet transfusions and Neupogen. He became very short of breath during the hospitalization. A specific cause was not identified. Bronchoscopy was done during the admission but nothing grew out. It was recommended that he not receive further Alimta. It was recommended that he receive a course of carboplatin and gemcitabine.

In September 2011, Mr. Kelly returned to Texas. He received the first course of chemotherapy with carboplatin and gemcitabine. He again developed cytopenia and was unable to receive day eight of the gemcitabine. He received the second course of

chemotherapy in October 2011. He again developed anemia requiring blood transfusions and did not receive day eight of the gemcitabine.

Since then he has not receive further chemotherapy. He has been followed up at the University of Texas Southwestern and at the University of Chicago. He had clinically stable disease as of May 2012. I have no information regarding Mr. Kelly's current clinical status.

Mr. Kelly clearly does not have a curable illness and will ultimately die of complications of the mesothelioma.

As I indicated in my report of December 12, 2011, the only known cause of malignant mesothelioma in man is prior asbestos exposure. Many studies have clearly demonstrated this relationship.

I had an opportunity to get a work history from Mr. Kelly and review the interrogatories. Both are summarized in my report of December 12, 2011. Mr. Kelly was exposed to asbestos while in the Navy. His naval experience is identified in my report. He also had some paraoccupational exposures to asbestos which are also described in my previous report.

My opinions remain the same as indicated in my prior report, a copy of which is enclosed.

Plaintiffs' Expert Designation

I am a physician currently practicing in Berkeley, California. I am a pulmonologist and critical care specialist. A pulmonologist is a physician who takes care of people with lung disease or who have disease involving other body parts that affect lung function; a critical care specialist takes care of people in an intensive care unit. I currently practice on the Alta Bates campus at Alta Bates-Summit Medical Center in Berkeley, California. All opinions herein and to be provided at the time of trial will be given with reasonable medical certainty.

I am part of a group of what is now 14 physicians. The Alta Bates-Summit Medical Center is an 800-bed hospital which is involved in providing all types of medical care: medicine, surgery, obstetrics, neonatal care, psychiatry, etc. The group that I am involved with runs the critical care units, the respiratory services at the hospital, the pulmonary function labs. The respiratory services portion delivers respiratory care for people who are hospitalized with a variety of lung diseases. In the pulmonary function laboratories we test individuals' lung function to see if it is normal or abnormal, and there are a variety of tests that we perform to assess such function.

I currently spend approximately half of my professional time treating patients.

My educational background is as follows: I went to undergraduate school at Cornell University where I majored in chemistry. I graduated from Cornell in 1964. I then attended medical school at the State University of New York, Downstate Medical Center in New York City. I graduated from medical school in 1968. From 1968 to 1969, I was an intern in internal medicine. Internal medicine is the study of diseases of all the body. My internship was at Michael Reese Hospital in Chicago from 1968 to 1969. I then became a resident of internal medicine at the University of Chicago Hospitals and at Michael Reese Hospital. From 1971 to 1974, I was at Stanford University where I did my training in pulmonary diseases and critical care.

From 1974 to 1976, I was in the U.S. Air Force. I was stationed in Illinois at the Air Force's worldwide center for lung disease. In that capacity, I saw patients with varying forms of lung disease and pulmonary complications/problems from all branches of the military and their dependents. If the patients were far from our center they were transported by an Air Force intensive care unit in the sky, in C-9's. I evaluated and managed patients with a huge variety of diseases.

Since 1976, I have been in practice in Berkeley at Alta Bates.

I am board certified in internal medicine and pulmonary diseases. To be board certified, you first have to be board qualified. Each area of medicine has a group of people who have determined 1) the appropriate training required for that given area of medicine and 2) the approved institutions one can be trained at for the specific area of medicine. Once an individual has undergone the appropriate training at the proper institution, each specialty gives examinations, depending on the specialty. If you pass the exam, you are then board certified and presumably competent to practice in the area or areas that you specialize in. I am board certified in internal medicine (1972) and pulmonary diseases (1975).

In 1976, as soon as I moved to Berkeley and began to practice medicine in private practice, I developed an interest in asbestos-related pulmonary diseases. The Northern California area around Berkeley, San Francisco and the general Bay Area contains an enormous number of people who were previously exposed to asbestos. This arose as a result of the industrial complexes, shipyards, refineries, and other building and industrial activities that occurred in the Bay Area during and after World War II. Many of these workers from our area developed pulmonary problems which wound up getting referred to our group.

In the normal course of my practice, I began seeing an enormous number of people with asbestos-related disease because either they had an abnormal x-ray and were referred to me or because they had symptoms like cough, shortness of breath or chest pain and their primary physicians were referring them for further evaluation. As a result of this source of patients suffering from the various asbestos diseases, I began to treat and diagnose people with asbestos disease.

The process of diagnosing individuals with asbestos disease includes taking work histories from people. This is a common and preferred practice among specialists in my area of medicine. It is important to determine an individual's whole asbestos exposure history in order to determine whether someone has a work-related illness. I therefore would ask from the first job to the last what they did. I would also inquire about other work practices that might have subjected them to asbestos exposure.

In order to familiarize myself with the exposures to asbestos that results from various work practices described by my patients, I have reviewed literature in the areas of medical and industrial hygiene regarding work practices that subject individuals to asbestos exposure. These documents have dealt with work practices involving asbestos gasket cutting, scraping and cleanup, insulation manipulation, asbestos cement work, packing, cutting, pulling and cleanup, brake blowout, brake grinding and sanding and brake cleanup, work with asbestos joint compounds, work with asbestos cement pipe, asbestos cement, etc.

From the beginning of my practice in the area of asbestos disease through today, I periodically review the literature regarding asbestos disease and work practices associated with an increased risk for asbestos disease. Initially this work was accomplished through research using the Index Medicus, which was published by the Library of Congress. It allows an individual to look up a topic in there and see what has been published all over the world that year on that topic. I would periodically review the literature on the topic of "asbestos" and follow the state-of-the-art for knowledge about the disease and the work practices that involve exposure which increased the risk for asbestos disease. In the last several years the information has been obtainable online. In addition to the medical and industrial hygiene literature, I also reviewed government publications regarding the position taken by the United States health and safety agencies and health and safety agencies from all over the world regarding the dangers posed by exposure to asbestos and causation of asbestos disease.

In my practice I have been involved in both the diagnosis and treatment of asbestos diseases. These include non-malignant asbestos diseases such as pleural plaques and asbestosis (scarring of the lung) and malignant diseases, for which the two main cancers caused by asbestos are lung cancer and mesothelioma. Lung cancer is a cancer that arises from the lung tissue itself. Mesothelioma is a cancer that arises from the lining of the lung. I have reviewed literature in terms of causation of mesothelioma as part of my treatment and diagnosis of many, many individuals over the course of my practice who suffered from that disease.

I have had an opportunity to review medical records, including pathology records, for hundreds of individuals diagnosed with mesothelioma. I have taken work histories from hundreds of individuals suffering from mesothelioma. I have determined how mesothelioma manifests itself in many dozens of individuals who I have treated and diagnosed with mesothelioma.

I have taught both physicians and medical students while on the faculty of the University of California, San Francisco Medical School. I also have worked for the Department of Labor regarding asbestos disease.

Starting in late 1977 and early 1978, two of my colleagues and I evaluated a group of workers who worked at the Hunters Point Naval Shipyard in San Francisco and/or the Mare Island Shipyard in Vallejo. We evaluated people who worked in the interiors of surface ships or submarines that were being constructed or repaired. Our study involved answering two questions: 1) what was the incidence of asbestos-related disease in this group of shipyard workers? and, 2) did it matter what you did at the shipyard in terms of what your risks were? Our findings from our study were published in the peer review literature.

The Department of Labor, which among other places, is based in San Francisco, became aware of the fact my colleagues and I were doing this study and were interested in asbestos-related disease. So in 1979, the Department of Labor began sending large numbers of federal civilian employees to one of my partners and I to evaluate for asbestos-related disease. Over the next 20 years, we probably saw approximately a couple thousand people for the Department of Labor to assess for work-related illness. The overwhelming majority of those we were evaluating were for asbestos-related disease.

This work on behalf of the federal government, was evaluating people for the potential that they would have some recovery based on their exposure to asbestos. Soon thereafter, my office began to perform the same duties for the State of California Worker's Compensation system, evaluating private employees to determine if there was a disability or disease related to asbestos exposure. Finally in around 1981, I began to testify in the third-party legal system as an expert witness. I have testified as an expert witness on behalf of Plaintiffs and Defendants. However, the vast majority of my work in recent years has been on behalf of Plaintiffs in asbestos-related litigation.

I also have significant experience in the administrative part of hospital work. I spent about fourteen years starting in the mid-1980's, at what was called medical director of clinical quality and resource management at my hospital. My job was to evaluate how to deliver excellent care to our patients in a climate in which revenue and financial resources were decreasing. I studied costs, particularly in relationship to technological advances becoming more prevalent in patient care. I became the physician representative in our hospital working with the administration and as part of a consortium all over the United States studying the costs of treatment. I have worked with Santa Monica Hospital, Cedars-Sinai Hospital, USC Hospital, UCLA Hospital and hospitals across the country. As part of this responsibility, I have become aware of care costs for treatment and how to review care costs in documents from around the country.

If I am the first medical witness, I expect that I would present what I call Lungs 101. This is a presentation about the anatomy of the chest and how the lungs work. The jury needs this in order to understand later specific testimony regarding Mr. Kelly. What I generally do is to present drawings on the anatomy of the chest, which comes from The Ciba Collection of Medical Illustrations prepared by Dr. Frank Netter. This was published in 1979. The drawings I generally use and can be found on pages 3, 4, 13, 23, 24, 28, and 29. These are pictures of the general anatomy of the chest, the chest wall, the airways, the pulmonary circulation, and the air sacs. I discuss the fact that the primary purpose of the lungs is to get oxygen into the body and carbon dioxide out of the body. I will point out that tiny asbestos fibers follow the same route as oxygen to get into the lungs. They deposit in the terminal bronchioles and air sacs and produce an inflammatory response.

The next issue that is typically discussed is what diseases are known to be caused by exposure to asbestos. This usually leads to a brief discussion of the nonmalignant diseases, that is, asbestos related pleural disease and asbestosis. Also typically discussed is the fact that exposure to asbestos causes malignancy, specifically, mesothelioma and lung cancer.

I expect that I would be asked about some general issues regarding mesothelioma. This includes the typical presentation of the disease. It also includes the natural history of the illness, including the symptoms that patients complain about late in the course of the disease. I have had considerable experience in seeing patients with mesothelioma and I am very familiar with its clinical presentation and clinical course.

I expect that I will be talking about what is known about the cause of mesothelioma. The only known cause of malignant mesothelioma in man is prior asbestos exposure or exposure to a similar substance called zeolite (in central Turkey). I will likely be asked about latency period. Mesothelioma is typically diagnosed decades after the onset of exposure to asbestos. The average latency period is 32 or 33 years with a wide distribution around that time interval. I do not think there will be any dispute regarding latency issues, but the jury needs to understand latency.

I also expect to discuss the fact that the risk for developing mesothelioma is dose dependent. The more asbestos in individual inhales the greater the risk for developing mesothelioma. Individuals very heavily exposed to asbestos are at very great risk for this otherwise rare disease. However, it is also known that even very low exposure to asbestos places individuals at risk for this disease. It is well known that individuals who have paraoccupational exposure to asbestos are at risk for mesothelioma. This was reported by Dr. Muriel Newhouse and an international symposium in New York City in 1964 and subsequently published in several places in 1965, including the British Journal of Industrial Medicine. Since then, there have been many studies published demonstrating that the risk of mesothelioma with paraoccupational exposure.

I expect that I will talk about Mr. Kelly's clinical presentation and course of disease. The information I will rely on is in this report. I will discuss the basis of the clinical decisions made by his treating physicians, including why he was placed on chemotherapy.

I expect to talk about causation as it specifically relates to Mr. Kelly.

There is no question that Mr. Kelly was exposed to asbestos as a shipyard worker. Shipyard workers or merchant seamen have been demonstrated to be at increased risk for mesothelioma. Kolonel et al, Cancer Res 45: 3924, 1985, Giarelli Am J Ind Med 22: 521, 1992, Varouchakis et.al. Am J Ind Med 19: 673, 1991.

I expect to be asked whether exposure to chrysotile asbestos causes malignant mesothelioma. I have concluded that exposure to chrysotile causes mesothelioma. This issue has been addressed by multiple United States governmental agencies and international scientific bodies. The Occupational Safety and Health Administration, the Consumer Product Safety Commission, the United States Agency for Toxic Substances and Disease Registry, the Environmental Protection Agency, the World Health Organization, and he International Agency for Research on Cancer have all concluded that exposure to chrysotile asbestos causes malignant mesothelioma.

In addition, there are multiple scientists in the United States who have written about this in the medical literature who have also expressed their opinion that exposure to chrysotile asbestos causes mesothelioma. A number of authors have addressed this issue including Lemen, Welch et al, Stayner et al, and Smith et al, Lemen, Int J Occup Environ Health 10:233, 2004; Welch et al, Int J Occup Environ Health 13:318, 2007; Stayner et al, Am J Pub Health 687, 1996; Smith et al, Am J Ind Med 33:94, 1998.

This issue has also been addressed in the Helsinki Criteria published in the Scand J Work Environ Health 23:311, 1997. This is a consensus report from scientists who also concluded that chrysotile exposure causes mesothelioma. There is no requirement in the Helsinki Criteria that there be a quantitative estimate of a person's dose of exposure exceeding a particular level in order to conclude that the mesothelioma was caused by prior asbestos exposure. There are also case control studies that have demonstrated that low levels of exposure increase the risk for mesothelioma; Rodelsperger Am J Indus Med 39:262, 2001, Iwatsubo et al, Am J Epidemiol 148:133, 1998.

I have also reviewed the literature on this issue and have concluded that chrysotile causes mesothelioma. First of all, chrysotile miners have been demonstrated to be at risk for mesothelioma. This has been demonstrated in the Canadian mines published in multiple papers; Begin et al, Am. J. Ind. Med. 22: 531, 1992, McDonald et al, Ann Occup Hyg 41: 707, 1997 as well as in chrysotile miners from Italy (Mirabelli, et al, Occup.Environ Med. 65: 815, 2008. Some investigators have expressed the opinion that it is the tremolite contamination of the chrysotile ore which is responsible for the mesothelioma in Canadian miners. However, the Italian mines are not contaminated with tremolite.

There is no amphibole contamination of the chrysotile ore in Italy; Turci et al, Ann Occup Hyg 53: 491, 2009.

A study was published in 2001 by Yano et.al. demonstrating mesothelioma in a factory in Western China. Only chrysotile was used in the factory and it was not contaminated with tremolite, Yano et al, Am J Epidemiology 15: 538, 2001.

A very important series of studies was published by Dr. William Nicholson at the Mount Sinai School of Medicine in New York City. Dr. Nicholson first studied insulators from New York and New Jersey. They were first exposed to amosite in the late 1930s when it began being put into insulation. He calculated when the insulators would be expected to get mesothelioma if only amosite exposure caused it and not chrysotile. He then had the actual data when the insulators actually got mesothelioma. These two curves were completely different. The insulators got mesothelioma much earlier than would have been expected if amosite exposure was the only cause. He therefore concluded that exposure to chrysotile causes mesothelioma. Dr. Nicholson then went on to do the same analysis for the entire 17,800 insulators in North America and came to the same conclusion. (Nicholson et al, Med Lav 86: 393, 1995, Nicholson Ind Health 39: 57, 2001.

He did a study of asbestos cement workers in Scandinavia and found similar results. A factory was studied where crocidolite was added to the mixture. The group got mesothelioma earlier than would have been expected if only exposure to crocidolite was the cause.

I expect that there will be several other important issues that may be brought up at trial. I have concluded that the work done by other military personnel with gaskets and packing contributed to his risk for the development of mesothelioma. I base this on data that it has been published in the literature demonstrating the release of free asbestos fibers during the use of gaskets and packing. I am relying on work published in the medical literature by McKinnery and Moore, Am Ind Hyg Assoc J 53: 531, 1992, Millette et al, EIS Technical Journal Fall 1995, Cheng and McDermott, Appl Occup Environ Hyg. 6: 588, 1991, and Longo et al, Applied Occ And Env Hyg 17: 55, 2002.

I also expect to talk about the costs of his care.

Finally, I may be giving testimony on "State of the Art" issues. This is regarding when it was known that exposure to asbestos causes asbestosis, lung cancer and mesothelioma. I have given testimony on this subject in the past and I expect that my testimony will be similar to what I have given in prior trials.

I am also including a copy of my most recent curriculum vitae. I have also provided you with a list of the depositions and court testimony that I have done over the last six years.

I charge $500.00 per hour for work done in my office, for deposition and for trial testimony.

Sincerely,

Barry R. Horn, M.D.

BRH:bh:dpf

Enclosures

CURRICULUM VITAE

Barry R. Horn, M.D.

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Born: May 4, 1942, New Rochelle, New York

EDUCATION AND TRAINING

1957 – 1960	New Rochelle High School, New Rochelle, New York
1960 – 1964	A.B. (with honors) Cornell University, Ithaca, New York
1964 – 1968	M.D., State University of New York, Downstate Medical Center, Brooklyn, New York
1968 – 1969	Intern - Straight Medicine, Michael Reese Hospital and Medical Center, Chicago, Illinois
1969 – 1970	First Year Resident in Medicine, Michael Reese Hospital and Medical Center, Chicago, Illinois
1970 – 1971	Second Year Resident in Medicine, University of Chicago Hospitals, Chicago, Illinois
1971 – 1972	Instructor in Physiology, Stanford University School of Medicine, Stanford, California
1971 – 1974	Research and Clinical Fellow in Respiratory Diseases, Stanford University School of Medicine, Stanford, California

STAFF POSITIONS

1973 – 1974	Medical Director, Division of Respiratory Therapy, Stanford Hospital, Stanford, California
1974 – 1976	Chief of Non-Tuberculous Pulmonary Disease, USAF Medical Center, Scott AFB, Illinois
1976 – 1986	Herrick Hospital and Health Center, 2001 Dwight Way, Berkeley, California
	Associate Director, Pulmonary Function Laboratory Associate Director, Intensive Care Unit Associate Director, Respiratory Unit Consultant in Pulmonary Diseases
1976 – Present	Assistant Clinical Professor, Department of Medicine, University of California Medical School, San Francisco, California
1977 – 1984	Pulmonary Consultant and Member of the Board of Directors, Western Institute for Occupational/Environmental Science, Inc.
1979 – 1987	Advisory Council Member, Bay Area Air Quality Management District; Chairman, Public Health Committee, 1980-83
1981 – 1987	Member of the Board of Directors, American Lung Association of Alameda County
1987 – Present	Alta Bates Medical Center, 2450 Ashby Avenue, Berkeley, California
	Consultant in Pulmonary Diseases Associate Director, Respiratory Therapy Associate Director, Pulmonary Function Laboratory Associate Director, Intensive Care Unit
1987 – 1995	Medical Director, Utilization Management
1995 – 1998	Medical Director, Clinical Quality and Resources Management

1997 – 1998	Vice President, Medical Staff, Alta Bates Medical Center, Berkeley, California
1997 – 1999	Trustee, Alta Bates Medical Center Board of Trustees
1997 – 1999	Member, Board Committee on Quality, Alta Bates Medical Center
1999 – 2001	President, Medical Staff, Alta Bates Medical Center, Berkeley, California
2000 – 2009	Trustee, Alta Bates-Summit Medical Center Board of Trustees
2000 – 2009	Member, Finance Committee of the Board of Trustees, Alta Bates-Summit Medical Center
2000 – 2004	Member, Board Committee on Quality Alta Bates-Summit Medical Center
2005 – 2009	Chairman, Board Committee on Quality Alta Bates-Summit Medical Center
2010 – 2011	Chairman, Board Committee on Quality Sutter Health East Bay Medical Center
2012 -	Member, Board Committee on Quality Sutter Health East Bay Medical Center
2008 – 2009	Secretary, Alta Bates Summit Medical Center Board of Trustees
2010 – 2011	Trustee, Sutter Health East Bay Medical Center
2010 – 2011	Chairman, Board Committee on Quality, Sutter Health East Bay Medical Center
2010 – 2011	Chairman, Medical Affairs Committee, Sutter Health East Bay Medical Center
2010 – 2011	Vice Chairman, Quality Committee, Sutter East Bay Medical Foundation

PROFESSIONAL MEMBERSHIPS

American Thoracic Society
American College of Chest Physicians
California Medical Association
Alameda-Contra Costa County Medical Association
California Thoracic Society

BOARD CERTIFICATIONS

Certified by the American Board of Internal Medicine, September 1972

Certified by the Pulmonary Disease Subspecialty Board, American Board of Internal Medicine, January 1975

Certified by the National Institute for Occupational Safety and Health as a Government-Certified AB≡ Reader for Interpretation of Chest Radiographs of Pneimoconioses

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BARRY R. HORN, M.D.
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11. Byrd, R.B., Horn, B.R., Griggs, G.A., and D.A. Solomon: Treatment of tuberculosis by the non-pulmonary physician. *Ann Int Med*, 86:799, 1977.

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13. Horn, B.R., and R.B. Byrd: Simulation of pleural disease by disk space infection. *Chest*, 74:575, 1978.

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15. Polakoff, P.L., Horn, B.R., and O.R. Scherer: The prevalence of radiographic abnormalities among Northern California shipyard workers. *Ann N.Y. Acad Sci*, 330:333, 1979.

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BARRY R. HORN, M.D.

2006 – December 2012 DEPOSITIONS & TRIAL TESTIMONY

2006 DEPOSITIONS

Samuel Gates
Forest Christian
Peter Jacobelly
Thomas Stroker
Kip Ong
Larry Brady
John Pisani
Troy Whittaker
Harold Roberts
Robert Hartford
Dana Barbour
Debra Hansen
Robert Johnson
Clair Miller
Lloyd Haanstra
Gregory Hartell
David Mosbacker
Alan Bergeson
Miriam Wilson
Thomas Halssema
Lawrence Campbell
Patrick White
Donald DeForge
John Miller
Saeed Behshid
Carl Pinson
Michael Robertson
Jack Reynolds
Allen Silver
Logus Adams
Paul Kraus
Micheli Santo
Richard Jones
Donald Collins
David Baake
Ventura Loza
Austin Frederick John
McNamara, John
Lupine
Robert Learn
Rebekah Price
Janet Warren
Ferrel Reid
Beverly Haegel
Bernard Sharp
Anthony Bergin
Rodney Smith
Christopher Pounds
Richard Fortini
George Leuenberger
Alan Spargo
Jacqueline Nunes
Islelore Essman
Dennis Morgen
Paul Whitlock
Allison Keith Hilton
Ronald Hewitt
James Ticer
Jerry Pinedo
Kenneth Sheffield
Kevin Trompeter
Susanne Delisle
Leslie Whiteley
Ernest Barber
Deborah Sue Huff
Charles Palin

2006 TRIAL TESTIMONY

Alan Reinstein
Lanny Horr
Rebecca Martinez
Thomas Stroker
Robert Johnson
Thomas Halsema
Richard Jones
George Barnes
David Baake
Rebekah Price
Paul Whitlock

2007 DEPOSITIONS

Charles Boyle
William Cileski
Gene Murray
David Vanderhyde
Ronald Redman
Ila Mae Sieberman
Philip Rincon
Paul Otto Shiebold
Ruben Flores
Richard Schultz
Ernest Ward
William Saller
Linda O'Donnell
Donald Eubanks
Norman Ford
Denzil Scott
Walter Rybaczyk
Fernando Jarueghi
Clayton Hogan
Emil Robles

Anthony Cueto
Billy Trout
Andres Ginez
Tony Ross
Robert Gianelli
Vieburan Leonard
Donald Felker
William Mackel
Edgar Hill
Daniel Murphy
Donald Farmer
Franz Losch
David Lathrop
Carl Stanglin
Edward McIntyre
Adrian Soper
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Linda O'Donnell
David Lathrop
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Pedro Barragan
Alvin Ehrig
Franklin Ham
Neil LeSage
Milton Stirm
John McTaggert
Daniel Mitchell
Alan Crowe
John Andregg
Vinton Champe
Earl Haupt
Bruce Cairns
Robert Griffiths
Harry Inman
Charles Bordeaux
Alfred Hall
Peter Daniels

2007 TRIAL TESTIMONY

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Don Felker
Neil LeSage

Fernando Jaruegui
Robert Learn
John Andregg
William Saller

2008 DEPOSITIONS

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Louis Gowan
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Terry Straughan

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Robert Carter
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Brenda Williams
Richard Orwig
Alan Nichols
Peter Galasi
Edward Phillips
Cornell Janes
Amanollah Shahabi
Dennis Hill

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Richard Dyhrman
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Odetta Hudiburgh
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Scott McGrail
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Jean Evans
Herbert Moreno
Adolph Tamayo

2008 TRIAL TESTIMONY

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Russell Roberts
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Paul Shiebold
Amanollah Shahabi
Leroy White
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Ulysses Collins
Larry Stewart
Franklin Yancey
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William Church
Wayne Miceli
Richard Buccola
Robert Kirchmann

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Parley Rey Worthen
Rhoda Evans
Dale Alan Rigg
Stanley Bjerke
Charles Cantrell
Patrick Fox
Roy DeHart
Norman Longstreet
Jerry Dyer
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Eva Reynolds
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Richard Spencer
James Prange
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Donat Lenney
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Paul Willoughby
William Calande
Napoleon Hall
Allen Hall
Howard Warren
John Casey
Thomas Lopez
Bennie Barnes
Vincent Amore
William Webb

2010 TRIAL TESTIMONY

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Stanley Bjerke
Donald Devore
Richard Cottrell
Arthur Strickland
Ronald Ricker
Gordon Bankhea

2011 DEPOSITIONS

Donald Osterberg
Duane Eastham
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Robert Gray
David Palomares
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Kenneth Grammer
Bobby Floyd
Luisa Nichols
Gerold Ricks x2
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Michael Bruno
Haskell Stillman
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Jesus Flores
Barbara Nine
Richard Sandoval
Benjamin Charlesworth
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Harry LeMaster
Robert Gottschall

William Harris
Jack Warren
Ronnie Pell
Vernest Hollowell
Thomas Metcalf x2
Michael Corbett
Aida Savelesky
Jack Olson
Ray Eversole
Doyle Green
Charles Smith
Ronald Kern
Richard Keeney
Mike Fidler
Timothy Vest
Robert Diana
Robert Brackett

2011 TRIAL TESTIMONY

John Casey
William Webb
Donald Osterberg
Donat Lenney
Billie Johnson
Robert Gray x2
William King x2
Michael Bruno
Francisco Nunez
Patricia DesBiens
Haskell Stillman
Henry Bissett
Mike Fidler

2012 DEPOSITIONS

Rick Fenstermaker
Ronald O'Branovich
Stanley Burden
Vernest Hollowell
Timothy Vest x2
Cornelius Murphy
Oscar Bonetto
Richard Lee
Joseph Oddo x2
Joseph Kosich
John Leonard x2
James Lovelace
Marc Anderson
Alan Martin
Plant Insulation
Clyde Norris
Thomas Acosta
Rudy Orona
John Swanson
Charles Smith
Alec Dieter
John Suitor
Clinton Bogan
Richard Miller
Nikki Pooshs
Alan Nichols
Marline Petitpas x3
Terry Clark
Joseph Wilcox
Thorpe Insulation
Landon McCuin
William Fox
Edward Carden
Harry Carpenter x2
Melvin Desin
Eugene Martin
Patrick Scott
Richard Luka
Sylvia Amundsen
George Patterson
Gene LePore
James David
Cheryl Benjamin
Kevin Stuckart
Daniel Skinner
James Hellam
Vincent Monaco
Raymond Bennett
Michael Corbett
Bobbie Holloway
Kurt Walter
Charles Denham
Raymond Smith
Julio Gomez x2
Michael Jordan
Nancy Easling

2012 TRIAL TESTIMONY

Richard Keeney
Richard Lee
Joseph Kosich
James Lovelace
Richard Lee
Marc Anderson
Joseph Oddo
John Leonard
Marline Petitpas x2
Plant Insulation
Moyer Hazelwood (VT)
John Suitor (VT)
Patrick Scott
Cheryl Benjamin
Melvin Desin
Daniel Skinner
Vincent Monaco
James David
James Hellam
Raymond Smith